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Haemodynamic and electrocardiographic accompaniments of resting postprandial angina[1, 2]

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SUMMARY The early postprandial changes in 10 patients with angiographically proven coronary artery disease and history of postprandial angina were studied by the continuous recording on magnetic tape of the electrocardiogram and haemodynamic variables. The significant changes 20 minutes after a meal not followed by angina included increases in cardiac index and stroke index, with a decrease in systemic vascular resistance. When angina developed after a meal, there were significant increases in mean systemic arterial blood pressure, heart rate, pulmonary capillary wedge pressure, and systemic vascular resistance with decreases in stroke index at the onset of pain rather than at the onset of ischaemic electrocardiographic abnormalities. The first haemodynamic variable to change was pulmonary capillery wedge pressure which tended to increase coincident in time with the electrocardiographic abnormalities. In all cases, postprandial angina occurred within 25 minutes after a meal. In every instance, there was little or no change in the product of heart rate and systolic arterial blood pressure at the onset of the ischaemic electrocardiographic abnormalities at a time when the pulmonary capillary wedge pressure had begun to rise. Postprandial angina, like many cases of rest angina, may arise on the basis of a primary decrease in myocardial perfusion, the nature of which is unclear but merits further investigation.

Angina occurring at rest but after a meal, is a well-known phenomenon in patients with advanced coronary artery disease (Cohen *et al.*, 1966; Friedberg, 1966; Harrison and Reeves, 1968). It generally occurs within the first 20 minutes after eating (Lewis, 1931; Wayne and Graybiel, 1933; Goldstein *et al.*, 1971), though occasionally it has been known to develop up to three to five hours later especially after a large fatty meal (Kuo and Joyner, 1955; Regan *et al.*, 1961). Furthermore, postprandial exercise tolerance is much impaired in these patients though a clear explanation for the phenomenon is not available (Wayne and Graybiel, 1933; Berman *et al.*, 1950; Goldstein *et al.*, 1971). It has been felt by some that myocardial ischaemia under these circumstances may be produced by an increase in oxygen demand related to postprandial increases in cardiac work (Friedberg, 1966; Harrison and Reeves, 1968; Goldstein *et al.*, 1971; Vatner *et al.*, 1970). However, an alternative mechanism involving primary coronary vasoconstriction caused by a reflex arising in the digestive tract (Wayne and Graybiel, 1933; Gilbert *et al.*, 1940; Morrison and Swalm, 1940), with a reduction in coronary blood flow (Kuo and Joyner, 1955; Regan *et al.*, 1961) has also been considered of significance in the development of postprandial angina.

An analysis of the haemodynamic changes preceding the onset of the ischaemic electrocardiographic abnormalities has been thought to be a useful tool for the indirect evaluation of the mechanism of angina (Guazzi *et al.*, 1971; Maseri *et al.*, 1975) and in a few instances changes in myocardial oxygen demand preceding the onset of angina have been reported. However, such sequential haemodynamic studies on resting postprandial angina have not been reported. This study was therefore undertaken to define the mechanism of postprandial angina from the records of various haemodynamic variables and the electrocardiogram obtained during

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the continuous monitoring of these functions on magnetic tape.

Patients and methods

Ten patients, eight men and two women, age range 46 to 82 years, were included in this study. All had a history of resting postprandial angina at home and while in hospital. They had coronary artery disease as indicated by a greater than 70 per cent obstruction of at least two major vessels documented by coronary angiography (seven patients), or by history of a previous myocardial infarction (three patients). All of them also had a history of exertional angina. All patients gave the history of postprandial angina as occurring within 20 to 25 minutes after a meal; the pain did not last longer than five minutes and it was readily relieved either spontaneously or by glyceryl trinitrate (GTN). In each instance acute myocardial infarction was subsequently ruled out by serial electrocardiographic and enzyme criteria. Patients with clinical or radiological evidence of heart failure or shock were not studied. Previous therapy except for sublingual GTN was discontinued at least 12 hours before the study. In some cases propranolol was tapered over a 48-hour period.

After written informed consent was obtained, arterial and pulmonary artery catheters were inserted and baseline measurements made. Arterial pressures and a modified lead of the electrocardiogram were continuously monitored while being displayed on an oscilloscope and recorded on magnetic tape. Cardiac output measurements were performed every two hours, and immediately before and 20 minutes after each meal. When angina supervened, cardiac output was also measured at the onset of the pain before the administration of GTN (if this was given) and when it was relieved. After stabilisation, all the haemodynamic variables and the electrocardiogram were recorded in every patient for varying periods before at least one meal and continuously thereafter for periods ranging between eight and 33 hours, average 21 hours. The continuous recording of the heart rate, arterial blood pressure, and the electrocardiogram permitted the delineation of the precise time relation in the alterations in these variables. Since the cardiac output could not be measured continuously, the changes occurring in this variable could only be evaluated with reference either to the last recorded measurement (often two hours previously) or to the one determined after the complete relief of pain under stable conditions. Each meal consisted of a mixed diet with a caloric content not exceeding 500 calories. On several occasions, the patents were fed by a nurse to facilitate the recording of haemodynamic variables. The patients were instructed to push an alarm button at the very onset of pain and to inform one of the investigators of the time of onset and complete relief of pain. A 12 lead electrocardiogram was taken at the beginning of the study, during pain, and after its relief. The procedure was approved by the Human Subjects Committee of the Cedars-Sinai Medical Center.

Arterial blood pressure was measured through a 21-gauge 2-inch long cannula inserted percutaneously into the radial artery. Pulmonary artery, pulmonary capillary wedge, and right atrial pressure were measured through a no. 7 Fr Swan-Ganz catheter inserted by cutdown into the antecubital vein, or percutaneously into the internal jugular vein. Cardiac output was measured in triplicate by the thermodilution technique (Ganz and Swan, 1972), and the arterial and pulmonary artery catheters were attached to a Stratham P23DB pressure transducer and Hewlett-Packard pressure amplifiers. The pressures and a modified lead V5 of the electrocardiogram were continuously displayed on an oscilloscope and recorded in a VR-3300 Data Tape magnetic tape recorder (Consolidated Electrodynamics). Both catheters were filled with a heparinised 5 per cent dextrose in water and flushed at least once every hour. The zero level was measured at 5 cm below the angle of Louis. Pressure measurements were taken by averaging all values over two respiratory cycles. At the end of the study the data stored on the magnetic tapes were recorded on a six-channel Clevite Brush Mark 200 ink recorder, at various paper speeds for the analysis of the overall patterns of change, as well as the beat-to-beat alterations.

Results

CLINICAL DATA

The clinical details of patients with respect to the extent of coronary artery disease, previous history of myocardial infarction, and treatment before the initiation of the study are shown in Table 1. Of the 10 patients, six experienced postprandial angina during the study. In all of them pain was promptly relieved by glyceryl trinitrate. The appearance of angina was not related to the amount of food, and in every instance it occurred within the first 25 minutes after the end of the meal. One patient developed angina 10 minutes after having eaten an apple, and another 15 minutes after a half-cup of cottage cheese.

HAEMODYNAMICS

The values of the measured haemodynamic variables in eight patients immediately before, and 15 to 20

minutes after finishing a meal not followed by pain, are shown in Table 2a. The measurements taken before the meal, and at the onset of postprandial pain and when the pain was relieved, in the six patients who experienced angina during the study, are shown in Table 2b. The trend in the haemodynamic changes was consistent in each of the three patients (cases 1, 5, 9) in whom more than one complete set of measurements with an without pain were available. The postprandial haemodynamic changes in the absence of pain indicated a significant ($P < 0.01$) decrease in systemic vascular resistance associated with a significant increase in cardiac index ($P < 0.01$) and stroke index ($P < 0.05$) without consistent changes in the heart rate (Fig. 1). The arterial pressure tended to decrease and there were no appreciable changes in pulmonary arterial, pulmonary capillary wedge, or right atrial pressure. In one patient (case 11), the resting pulmonary capillary wedge was abnormally raised in the absence of clinical or radiological signs of heart failure. This patient had had three myocardial infarctions in the past. The postprandial haemodynamic change in this patient was suggestive of a relative improvement in left ventricular function as indicated by an increase in stroke index and a

Table 1 *Clinical features of patients studied*

Case no.	*Sex*	*Age (y)*	*History of myocardial infarction*	*Previous therapy*	*No. of vessels involved (angio)*
1	M	82	1	Propranolol 80 mg; nitrates	—
2	M	73	2	Propranolol 80 mg	—
3	F	71	—	Nitrates	3
4	M	60	—	—	3
5	M	62	—	Propranolol 40 mg; nitrates	3
6	F	52	1	Propranolol 240 mg; nitrates	—
7	M	69	—	Propranolol 80 mg; nitrates	3
8	M	46	1	Propranolol 240 mg; nitrates	2
9	M	62	—	Nitrates	2
10	M	65	3	Nitrates	3

Table 2a *Postprandial haemodynamic changes in patients with advanced coronary artery disease in absence of chest pain*

Case no.		*HR (beats/min)*	*Systemic arterial pressure (S/D) (mmHg)*	*$\overline{AP}$ (mmHg)*	*Systemic arterial pressure $\times HR \times 0{\cdot}001$*	*PA pressure (S/D) (mmHg)*	*PCWP (mmHg)*	*RAP (mmHg)*	*CI (l/min per m²)*	*SI (ml/beat per m²)*	*SWI (gm/beat per m²)*	*SVR (dynes/s per cm⁻⁵)*
1	B	72	135/52	80	9·7	16/6	5	4	1·95	27·1	27·6	1888
	A	80	110/45	67	8·8	12/4	4	4	2·15	26·9	23·0	1420
2	B	58	142/54	83	8·2	32/12	13	5	2·06	35·5	33·8	1793
	A	68	130/48	75	8·8	37/14	14	5	2·24	32·9	27·3	1478
4	B	81	130/66	87	10·5	20/7	7	5	2·87	35·4	38·5	1299
	A	75	130/60	83	9·8	18/8	7	6	3·02	403	41·7	1164
5	B	68	135/65	88	9·2	32/13	12	6	2·77	40·7	42·1	1250
	A	64	138/66	90	8·8	34/14	11	6	3·11	48·6	52·2	1152
6	B	82	130/65	87	10·7	22/10	9	3	2·10	25·6	27·2	1926
	A	83	109/58	75	9·0	22/10	10	3	2·30	27·7	24·5	1512
8	B	62	103/56	72	6·4	21/7	6	5	2·00	32·3	29·0	1279
	A	67	104/54	71	7·0	23/6	5	3	2·90	43·3	38·9	892
9	B	55	127/60	82	7·0	20/8	8	5	2·12	38·5	38·7	1630
	A	53	132/60	88	7·0	23/9	9	6	2·17	40·9	43·9	1699
10	B	90	125/90	102	11·3	43/24	23	10	2·47	27·4	29·4	1716
	A	90	120/85	97	10·8	43/23	20	10	3·17	35·2	36·9	1256
M ± SEM	B	71·0 ±4·4	128·4 ±4·1/ 63·5 ±4·2	85·1 ±3·0	9·1 ±0·6	25·8 ±3·2/ 10·9 ±2·1	10·4 ±2·1	5·4 ±0·7	2·29 ±0·13	32·8 ±2·0	33·3 ±2·1	1598 ±100
	A	72·5 ±4·2	121·6 ±4·5/ 60·3 ±4·4	80·8 ±3·7	8·8 ±0·5	26·5 ±3·7/ 11·0 ±2·1	10·0 ±1·8	5·4 ±0·8	2·63 ±0·16‡	37·0 ±2·7†	36·1 ±3·6	1322 ±90*

A, before meal; B, after meal; HR, heart rate; $\overline{AP}$, mean arterial pressure; S/D, systolic/diastolic; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; CI, cardiac index; SI, stroke index; SWI, stroke work index; SVR, systemic vascular resistance. *$P < 0{\cdot}01$; †$P < 0{\cdot}05$; ‡$P < 0{\cdot}02$.

Table 2b *Haemodynamic changes during postprandial angina*

Case no.	HR (beats/min)	Systemic arterial pressure (S/D) (mmHg)	$\overline{AP}$ (mmHg)	Systemic arterial pressure ×HR× 0·001	PA pressure (S/D) (mmHg)	PCWP (mmHg)	RAP (mmHg)	CI (l/min per m²)	SI (ml/beat per m²)	SWI (g m/beat per m²)	SVR (dynes/s per cm⁻⁵)
1 B	68	168/74	105	11·4	26/11	11	5	2·20	32·4	41·4	2204
CP	87	184/86	119	16·0	52/26	25	6	2·15	24·7	31·0	2554
R	81	164/70	101	13·3	32/14	12	5	2·22	27·4	38·4	2180
2 B	62	155/54	88	9·6	40/15	15	5	2·07	33·4	33·2	1897
CP	120	192/89	123	23·0	70/36	38	11	2·22	18·5	20·9	2347
R	64	132/45	74	8·4	28/10	12	2	2·07	32·3	27·2	1646
3 B	92	174/58	97	16·0	36/16	8	6	2·85	31·0	37·5	1462
CP	105	240/120	160	25·7	52/34	32	12	—	—	—	—
R	94	180/64	103	16·9	30/11	10	5	—	—	—	—
4 B	66	135/70	92	8·9	24/9	7	5	2·62	39·7	45·9	1555
CP	98	150/90	110	14·7	54/30	28	6	2·41	24·5	27·3	2180
R	70	160/90	113	11·2	28/13	10	5	2·61	37·3	52·2	1982
5 B	72	115/58	77	8·3	36/13	12	7	2·87	39·9	35·3	1016
CP	92	160/84	109	14·7	54/34	28	10	2·89	31·4	34·6	1485
R	79	153/80	104	12·1	22/10	8	6	2·44	30·9	40·3	1508
7 B	83	135/67	90	11·2	24/10	8	4	2·23	26·9	30·0	1719
CP	87	150/70	97	13·0	30/16	14	6	2·40	27·6	31·2	1733
R	84	125/66	86	10·5	20/10	7	4	2·00	23·8	25·6	1829
B	73·8 ±4·7	147·0 ±9·2/ 63·5 ±3·2	91·5 ±3·8	10·9 ±1·1	31·0 ±2·9/ 12·3 ±1·1	10·2 ±1·2	5·3 ±0·4	2·47 ±0·14	33·9 ±2·1	37·2 ±2·9	1642 ±165
CP	98·2 ±5·2†	179·3 ±14·1‡/ 89·8 ±6·7†	119·7 ±8·9‡	17·9 ±2·1*	52·0 ±5·2**/ 29·3 ±3·0¶	27·5 ±3·3§	8·5 ±1·1‡	2·41 ±0·13	25·3 ±2·1†	29·0 ±2·3	2059 ±197†
R	78·7 ±4·3	152·3 ±8·4/ 69·2 ±6·3	96·8 ±5·8	12·1 ±1·2†	26·6 ±1·9§/ 11·3 ±0·7§	9·8 ±1·0¶	4·5 ±0·6†	2·27 ±0·11	30·3 ±2·3	36·7 ±4·8	1829 ±119

B, before meal; CP, during chest pain; R, during relief; † $P < 0.05$; ‡ $P < 0.02$; * $P < 0.01$; ** $P < 0.005$; § $P < 0.002$; ¶ $P < 0.001$.
Further abbreviations see Table 2a.

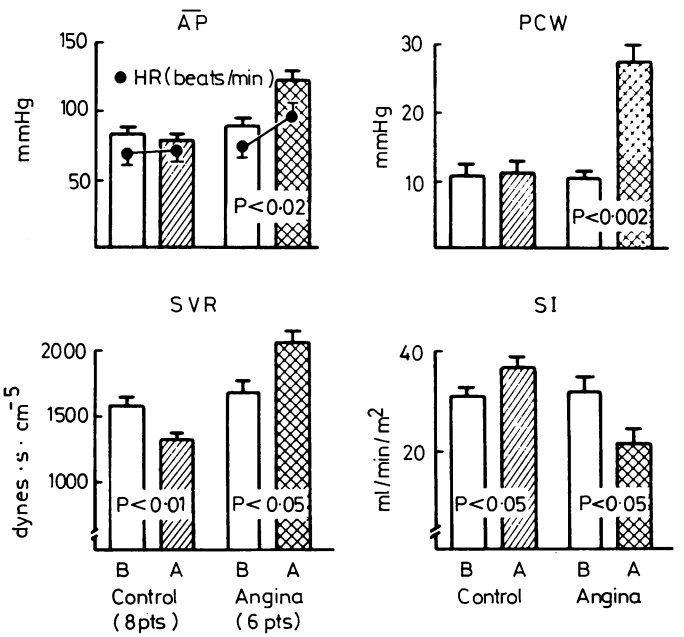

Fig. 1 *Mean postprandial haemodynamic changes with and without the occurrence of angina in patients with ischaemic heart disease. $\overline{AP}$, mean arterial pressure; PCW, mean capillary wedge pressure; SVR, systemic vascular resistance; and SI, stroke volume index. In each panel* B *represents mean values before the ingestion of a meal and* A *represents figures for the corresponding variables after a meal. Note that in the absence of angina, SVR falls and SI increases significantly with no change in PCW. During episodes of angina postprandially, there are significant increases in SVR and PCW, $\overline{AP}$ associated with a fall in SI.*

moderate decrease in pulmonary capillary wedge.

When postprandial angina occurred, however, there was a uniform increase in pulmonary arterial, pulmonary capillary wedge, heart rate, and arterial pressure (Fig. 1). The stroke index decreased and the systemic vascular resistance increased in four of the five patients in whom they were measured, and remained unchanged in the other patients (Fig. 1). The initial changes in pressures and heart rate, in every instance, were present before the onset of pain for an average period of three minutes (range one to eight minutes) (Fig. 2). The increases in heart rate, pulmonary arterial pressure, and arterial pressure were virtually simultaneous in all patients. In general, there was a further increase in these variables after the onset of pain (Fig. 2). Compared with episodes of resting angina not associated with meals it was noted that changes in pulmonary arterial pressure, stroke index, and arterial pressure were similar to those observed during postprandial angina. The increases in heart rate were, however, more striking during postprandial angina than those in spontaneous angina (Fig. 3).

The electrocardiographic abnormalities indicative of ischaemia, ST depression greater than 1 mm in five patients, and ST elevation greater than 1 mm in another patient, developed at the onset of haemodynamic changes preceding or following them by intervals no longer than 60 seconds.

Except for one patient (case 4), in whom angina was relieved spontaneously (Fig. 3), all patients obtained prompt relief with glyceryl trinitrate administered sublingually. At the point of total pain relief, changes in heart rate, arterial pressure, and pulmonary arterial pressure were reversed. Electrocardiographic abnormalities reverted to those present during the pre-anginal phase.

Discussion

The most prominent haemodynamic changes found in this study in patients with documented coronary artery disease were significant increases in cardiac output and decreases in systemic vascular resistance about 20 minutes after a meal in the absence of pain. There was also a tendency for the arterial blood pressure to decrease and for the heart rate to increase, and though these changes were not statistically significant, they were similar to the postprandial haemodynamic changes previously reported in normal subjects (Jones *et al.*, 1965), non-cardiac patients (Norryd *et al.*, 1975) as well as in patients with coronary artery disease (Wayne and Graybiel, 1933; Klakeg *et al.*, 1955; Goldstein *et al.*, 1971). The moderate albeit inconsistent increases in heart rate and cardiac output may constitute part of an increased sympathomimetic activity associated with the ingestion of food as has been postulated by some investigators who noted the absence of such a

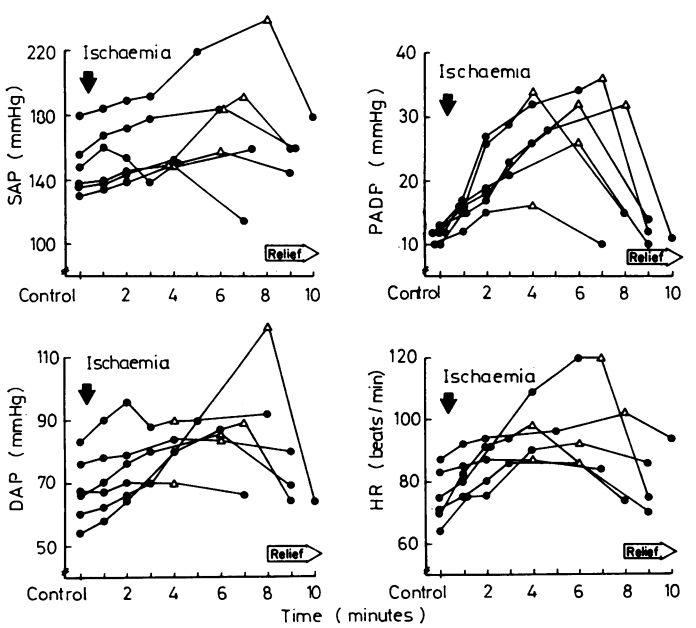

Fig. 2 *The time course of changes in SAP (systemic systolic arterial pressure), DAP (systemic diastolic arterial pressure), PADP (pulmonary artery end-diastolic pressure), and HR (heart rate) in 6 patients during episodes of postprandial angina. Ischaemic changes on the electrocardiogram occurred coincident in time with rises in PADP; changes in other haemodynamic variables supervened somewhat later in relation to the electrocardiographic abnormalities. Though not shown in the figure, the onset of pain invariably* followed *the development of the ischaemic changes.*

response in catecholamine-depleted dogs (Fronek and Stahlgren, 1968). A postprandial increase rather than a decrease or no change in arterial blood pressure has been noted previously in normal subjects (Grollman, 1929; Gladstone, 1935; Dagenais *et al.*, 1966) and in patients with coronary artery disease (Morrison and Swalm, 1940; Berman *et al.*, 1950). It should be emphasised, however, that in these studies measurements were taken at least 45 to 60 rather than 20 minutes after the completion of a meal, which, in addition, had a very much greater caloric content.

The increases in cardiac output found in our patients were associated with a pronounced fall in systemic vascular resistance consistent with peripheral vasodilatation. Such vasodilatation probably involves a number of vascular beds related perhaps to the release of a vasoactive hormone (Norryd *et al.*, 1975); the postprandial increases in flow to different areas such as the liver, kidneys, skin, and especially the splanchnic circulation have, in fact, been documented (Abramson and Fierst, 1941; Brandt *et al.*, 1955; Reininger and Sapirstein, 1957). The increase in cardiac output in our patients was accomplished essentially by an increase in stroke volume which, in the absence of an associated increase in left ventricular filling pressure, is suggestive of an increase in myocardial contractility, though it may simply be the result of a decrease in systemic vascular resistance, the nature of which remains unclear. However, the possibility must also be considered that an augmented venous return may not always be reflected in an increased left ventricular filling pressure if the heart size also increased, a mechanism that could account for the observed changes in cardiac output and stroke. An increase in contractility may also be affected by rises in the levels of serum glucose and fatty acids which, however, were not measured in our study. The relative roles of these various potential mechanisms cannot therefore be evaluated.

In our patients, postprandial angina was associated with haemodynamic changes which differed very much from those occurring in the same patient at the corresponding time after a meal in the absence of angina. During postprandial angina there was an increase in arterial blood pressure, right atrial pressure, and heart rate with striking increases in

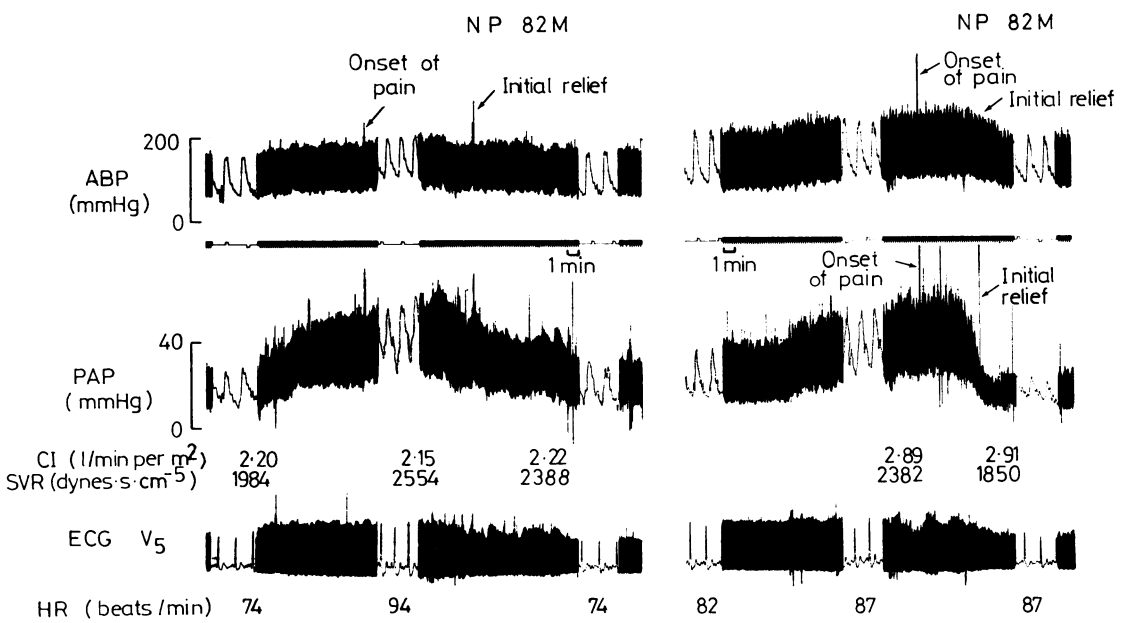

Fig. 3 *Haemodynamic and electrocardiographic recordings during two episodes of postprandial angina in a patient with known ischaemic heart disease. ABP, arterial blood pressure; PAP, pulmonary arterial pressure; CI, cardiac index; SVR, systemic vascular resistance; and HR, heart rate. Note that PAP began to increase before there were changes in HR or ABP and before the onset of pain. Increases in HR, ABP, and SVR occurred with the onset of pain. The panel on the left shows the time course of spontaneous relief of pain; that on the right shows the termination of the episode by glyceryl trinitrate administration.*

pulmonary arterial pressure and pulmonary capillary wedge, and a tendency for the cardiac output to decrease and for the systemic vascular resistance to increase. The concomitant increases in pulmonary capillary wedge pressure and decreases in stroke volume, also encounted in a parallel study in unprovoked or rest angina (Figueras *et al.*, 1978), as well as in exertional angina (Müller and Rørvik, 1958; O'Brien *et al.*, 1969; Lecerof, 1974) are indicative of an acute left ventricular failure. The increases in arterial blood pressure occurred immediately after or at the onset of the ischaemic electrocardiograph abnormalities suggesting that they were more likely to be the result rather than the cause of myocardial ischaemia. In line with this, previous observations in resting angina by us and others (Guazzi *et al.*, 1975; Figueras *et al.*, 1978), as well as in exercise-induced angina (Kattus *et al.*, 1975), have indicated that arterial blood pressure may increase sharply during ischaemia before the onset of pain. Vasopressor reflexes originating from the ischaemic myocardium observed under experimental conditions have been implicated as a possible explanation for these increases in arterial blood pressure (Vatner *et al.*, 1970) but the significance of such reflex activity in man is unclear.

It is of interest that despite the rather low caloric intake and variable composition of the meals, the time of onset of ischaemia in our own studies in man was similar in all the patients studied, as well as in 30 other patients with postprandial angina in whom the study could not be performed. In this respect our findings confirm previous studies that indicated that neither the amount nor the content of the ingested food had significant bearing on the appearance of postprandial angina (Wayne and Graybiel, 1933; Klakeg *et al.*, 1955). Though it has been claimed that, specifically, the ingestion of a large fatty meal is responsible for postprandial angina in some patients, it appears to occur significantly (3–5 hours) later, and it apparently coincides with the peak levels of the plasma triglycerides (Kuo and Joyner, 1955; Friedman *et al.*, 1964). Decreased coronary blood flow by the increased plasma viscosity has been suggested as the mechanism of ischaemia in these cases (Kuo and Joyner, 1955; Regan *et al.*, 1961).

It is now increasingly recognised that many patients with coronary artery disease experience a rest angina unrelated to meals or other precipitating causes; the mechanism here may be related, at least on inferential evidence, to spontaneous decreases in coronary blood flow (Gorlin, 1965; Guazzi *et al.*, 1975; Figueras *et al.*, 1978). The present study suggests that a primary reduction in coronary blood flow may also be responsible for angina that follows meals in patients with coronary artery disease. For example, clear differences were found in the haemodynamic alterations in the postprandial phase in the absence of chest pain and those during postprandial angina in the same patients. Of particular significance was the observation that the double product (SAP $\times$ HR) as an index of MVO_2 (Braunwald, 1971), though enhanced after the onset of pain, was altered little or inconsistently *at the onset* of the ischaemic electrocardiographic abnormalities, indicating that an augmentation in the oxygen demand was unlikely to be the primary mechanism of angina in this context. Though our data raise the possibility that postprandial angina may arise on the basis of a primary reduction in coronary blood flow, the nature of such a reduction in myocardial perfusion remains unknown. Coronary vasoconstriction may represent a potential mechanism and it is of interest that gastric distension has been suggested as a possible source of a coronary vasoconstrictor reflex in patients with postprandial angina (Gorlin, 1965). The possibility must also be considered that the development of angina after meals may represent a coincidental occurrence of rest angina, related to intermittent primary alterations in myocardial perfusion in patients with coronary artery disease. Investigations with measurements of myocardial perfusion in patients in whom angina can be produced predictably and repeatedly by a meal are thus likely to provide a more definitive mechanism of postprandial angina.

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